

Public Health Goal for LEAD in Drinking Water

Prepared by

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PREFACE

Drinking Water Public Health Goal of the Office of Environmental Health Hazard Assessment

This Public Health Goal (PHG) technical support document provides information on health effects from contaminants in drinking water. The PHG describes concentrations of contaminants at which adverse health effects would not be expected to occur, even over a lifetime of exposure. PHGs are developed for chemical contaminants based on the best available toxicological data in the scientific literature. These documents and the analyses contained in them provide estimates of the levels of contaminants in drinking water that would pose no significant health risk to individuals consuming the water on a daily basis over a lifetime.

The California Safe Drinking Water Act of 1996 (amended Health and Safety Code, Section 116365) requires the Office of Environmental Health Hazard Assessment (OEHHA) to adopt PHGs for contaminants in drinking water based exclusively on public health considerations. The Act requires OEHHA to adopt PHGs that meet the following criteria:

1. PHGs for acutely toxic substances shall be set at levels at which scientific evidence indicates that no known or anticipated adverse effects on health will occur, plus an adequate margin-of-safety.
2. PHGs for carcinogens or other substances which can cause chronic disease shall be based solely on health effects without regard to cost impacts and shall be set at levels which OEHHA has determined do not pose any significant risk to health.
3. To the extent the information is available, OEHHA shall consider possible synergistic effects resulting from exposure to two or more contaminants.
4. OEHHA shall consider the existence of groups in the population that are more susceptible to adverse effects of the contaminants than a normal healthy adult.
5. OEHHA shall consider the contaminant exposure and body burden levels that alter physiological function or structure in a manner that may significantly increase the risk of illness.
6. In cases of scientific ambiguity, OEHHA shall use criteria most protective of public health and shall incorporate uncertainty factors of noncarcinogenic substances for which scientific research indicates a safe dose-response threshold.
7. In cases where scientific evidence demonstrates that a safe dose-response threshold for a contaminant exists, then the PHG should be set at that threshold.
8. The PHG may be set at zero if necessary to satisfy the requirements listed above.
9. OEHHA shall consider exposure to contaminants in media other than drinking water, including food and air and the resulting body burden.
10. PHGs adopted by OEHHA shall be reviewed periodically and revised as necessary based on the availability of new scientific data.

PHGs adopted by OEHHA are for use by the California Department of Health Services (DHS) in establishing primary drinking water standards (State Maximum Contaminant Levels, or MCLs). Whereas PHGs are to be based solely on scientific and public health considerations without regard to economic cost considerations, drinking water standards adopted by DHS are to consider economic factors and technical feasibility. For this reason PHGs are only one part of the information used by DHS for establishing drinking water standards. PHGs established by

OEHHA exert no regulatory burden and represent only non-mandatory goals. By federal law, MCLs established by DHS must be at least as stringent as the federal MCL if one exists.

PHG documents are developed for technical assistance to DHS, but may also benefit federal, state and local public health officials. While the PHGs are calculated for single chemicals only, they may, if the information is available, address hazards associated with the interactions of contaminants in mixtures. Further, PHGs are derived for drinking water only and are not to be utilized as target levels for the contamination of environmental waters where additional concerns of bioaccumulation in fish and shellfish may pertain. Often environmental water contaminant criteria are more stringent than drinking water PHGs, to account for human exposures to a single chemical in multiple environmental media and from bioconcentration by plants and animals in the food chain.

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SUMMARY

A Public Health Goal (PHG) of 2 ppb is developed for lead in drinking water based on the neurobehavioral effects of lead in children and the hypertensive effects of lead in adults. Lead has been known as a toxic metal since ancient times, and has been studied for its toxic effects since the middle ages. It is a widespread contaminant in the human environment and occurs in drinking water as a consequence of leaching from plumbing containing lead. Lead may also enter drinking water from other sources, for example, directly from soil contaminated with lead or deposited from air emissions. Lead has multiple toxic effects on the human body. Among its most serious noncarcinogenic effects are decreased intelligence in children and increased blood pressure in adults. Lead is a carcinogen in laboratory animals and a probable human carcinogen. The U.S. Environmental Protection Agency (U.S. EPA) has adopted a Maximum Contaminant Level Goal (MCLG) of zero for lead in drinking water based on "occurrence of low level effects" and because U.S. EPA classifies lead as a Class B₂ carcinogen. Based on the toxicological data for noncarcinogenic endpoints, and potential human exposures, a PHG of 0.002 mg/L (2 ppb) is calculated for lead in drinking water.

INTRODUCTION

The purpose of this document is to develop and propose a PHG for lead in drinking water. Lead may be present in surface water or ground water sources, but more frequently it enters tap water in the distribution systems, often at the individual home. Lead pipes and solder corrode and leach lead into tap water. Tap water is used for drinking directly and also for the preparation of foods and beverages. Lead has toxic effects on all systems of the body, particularly on the gastrointestinal tract, hematopoietic system, kidneys and central nervous system.

CHEMICAL PROFILE

Lead is a metallic element, the 82nd element on the periodic table. Naturally occurring lead has an average atomic weight of 207.2. It is made up mainly of three stable isotopes with atomic weights of 208 (52%), 206 (26%) and 207 (21%). There are also naturally occurring radioactive isotopes of lead. Small amounts of lead are produced by the decay of heavier radioactive elements, both natural and synthetic (CRC Handbook of Chemistry and Physics, 1994). Lead is a bluish gray or gray-white metal with a bright silvery luster. It is very soft, highly malleable and ductile. Lead is a poor conductor of electricity, and is resistant to corrosion.

The melting point of metallic lead is 327.4°C; its boiling point is 1,740°C. The density of metallic lead is 11.34 g/cm³ at 20°C. Metallic lead is soluble in nitric or sulfuric acid, but insoluble in water or organic solvents. Lead salts such as lead nitrate and lead acetate are soluble in water. The usual valence states of lead are 0, +2 and +4. Lead can easily be alloyed with antimony, tin or other metals. Common lead salts include: acetate, chloride, chromate, nitrate, oxide, phosphate and sulfate. Lead can also be part of organic compounds, and can be chelated by various ligands (CRC Handbook of Chemistry and Physics, 1994).

Lead is easily obtained from its most common ore, galena (PbS). Because lead is so easily obtained, it was one of the first used metals, and remains one of the most used metals. Lead is mentioned in the book of Exodus, and other ancient literature. The many commercial uses of lead follow from the physical and chemical properties described above. For its malleability and

workability lead is used in piping, roofing and other structural uses. Because of its resistance to corrosion it is very long-lasting; some ancient Roman water pipes are still in use. Lead is also used in making containers for corrosive liquids. Metallic lead and lead dioxide are used in storage batteries for automobiles and other applications. In the past, organolead compounds were used to boost octane (reduce knock) in gasolines, but this use has been eliminated in California. Lead and lead salts have been widely used in paints and pigments, and in glazes for ceramics. Cable coverings are made from lead because of its electrical resistance and ductility. Lead is used to make bullets and shot. Because of its low melting point lead is used (with other metals) to make solder. Lead is used for radiation shielding around diagnostic x-ray machines and other sources of radiation (CRC Handbook of Chemistry and Physics, 1994; U.S. EPA, 1986).

In the past lead was included in a number of medicines such as antiseptics and astringents, but these are no longer recommended because of the cumulative toxic effects of lead in the body.

ENVIRONMENTAL OCCURRENCE

Lead is widely distributed in the environment. It is found in all media including air, water, food and soil.

Air

The use of tetraethyl lead as a gasoline additive resulted in widespread air pollution. Over the last two decades, the amount of lead in the air has been greatly reduced by the introduction of unleaded gasolines (OEHHA, 1997). Leaded gasolines are still widely used in many other countries.

Smelters and refineries emit lead into the air. Lead in contaminated soil becomes airborne when soil particles are picked up by the wind, or when soil is disturbed by digging, grading, plowing or gardening. The average level of lead in ambient air in California is 0.04 to 0.06 $\mu\text{g}/\text{m}^3$. Most of this lead is in the form of lead-containing particles (OEHHA, 1997).

Soil

Contamination of soil by lead is widespread in California and elsewhere. Lead has been deposited in soil in a number of ways. Much of the lead in soil came from the combustion of leaded gasoline. Lead from lead paint has also been deposited in soil, particularly around older homes. Land disposal of lead storage batteries has also contributed to soil contamination in many sites. Some of these lead storage battery disposal sites have very high levels of lead contamination, up to a few percent of the soil.

A national survey of soil lead in the United States (U.S.) found levels that ranged from 10 ppm to 700 ppm, with an average of about 15 ppm (Shacklette *et al.*, 1971). Fifteen parts per million has also been given as the average naturally occurring soil lead level (Lovering, 1976). The California Department of Health Services (DHS) conducted a survey of residential soil lead levels in parts of

three major cities suspected to have high lead concentrations: Oakland, Sacramento and Los Angeles (DHS, 1991). The median residential soil lead levels for these three cities were:

Oakland	880 ppm
Sacramento	230 ppm
Los Angeles	190 ppm

In all three cities there were some household soil samples with thousands of parts per million of lead.

Water

Environmental bodies of water become contaminated from contact with soil. In addition, drinking water becomes contaminated in the distribution systems because of lead in plumbing and solder. Lead enters drinking water from lead in pipes and and fixtures and from lead solder used to join pipes (Mahaffey, 1985). This is particularly troublesome in older homes. In newer homes the use of lead in pipes and solder has been greatly reduced or eliminated (Mahaffey, 1985). In addition to homes, older public buildings such as schools and theaters may also have problems with lead contamination of drinking water (Mahaffey, 1985).

According to Pocock *et al.* (1983), "Individual blood lead can be considerably increased by raised household tap water lead concentrations." After studying middle-aged men in 24 British towns, they estimated that the mean blood lead level is 43% higher for men when the concentration of lead in first-draw domestic tap water is 100 µg/L compared with lead-free water. This study demonstrates that tap water lead may be a significant source of exposure for some populations.

PHARMACOKINETICS

When lead is ingested in drinking water or foods, a fraction of it is absorbed into the bloodstream via the gastrointestinal tract. Lead in the bloodstream becomes deposited in tissues, mainly in bone. Blood lead is also excreted via the feces and urine.

Absorption

Children absorb more lead than do adults (Ragan, 1983). This has been clearly demonstrated in a number of studies. Absorption of lead into the blood from the gastrointestinal tract appears to be low in human beings compared to animals, although it is higher in children than in adults (Ragan, 1983). This is one reason why children are more sensitive than adults to lead exposure by the oral route. Children absorb about 40 to 50% of ingested lead, whereas adults absorb only at 5 to 15% (Ragan, 1983).

Heard and Chamberlain (1982) performed experiments with eight adult male volunteers who ingested ²⁰³Pb in the presence and in the absence of calcium and phosphate. With no minerals present, uptake averaged 63.3%. The two minerals together (200 mg Ca and 140 mg phosphate) reduced this to 10.6%, but neither mineral by itself was nearly as effective as the two together.

James *et al.* (1985) estimated absorption of lead from drinking water for adults at 2 to 10% during meals, and 40 to 60% between meals. O'Flaherty (1993) suggested a value of 8% based on a review of the literature, and demonstrated that this absorption factor works well for predicting

adult blood lead levels from exposure via drinking water and food. Bowers *et al.* (1994) have derived an uptake slope factor of 0.037 µg/dL blood per µg/day based on data correlating adult blood lead with lead in household drinking water.

Distribution

Once lead is absorbed into the bloodstream it becomes distributed throughout the body. It is deposited in various tissues including the bones, kidneys and central nervous system. Long-term storage of lead is mainly in the bones.

Lead which is absorbed from the gastrointestinal tract enters the blood. The lead which is not eliminated in the urine or feces is distributed into the tissues of the body including the bone, brain and kidneys (Rabinowitz, 1991). The residence time of lead in the soft tissues (brain and kidneys) is much shorter than in the bone. Lead accumulates in the bone with time, and lead levels in the bone generally increase with age (Rabinowitz, 1991). The storage of lead in bone depends on the diet; higher levels of calcium and iron in the diet tend to protect against deposition of lead into the bone (Rabinowitz, 1991; Silbergeld, 1991). During pregnancy lead is often remobilized from bone and may be transferred from mother to fetus (Silbergeld, 1991). High blood lead levels may indicate recent exposure, or in some cases they may reflect remobilization of lead from bone storage (Silbergeld, 1991).

Rothenberg *et al.* (1994) studied blood lead levels of women during normal pregnancies in Mexico City. They found that the women's blood lead levels decreased during the period from the 12th to 20th week of pregnancy, but increased almost linearly from the 20th week to delivery. It remains to be determined whether this pattern of decrease followed by increase is general; and if it is general, it remains to be explained (Rothenberg *et al.*, 1994).

Excretion

Absorbed lead is excreted mainly through the urine, but also in the bile and to a small extent in sweat, hair and fingernails (Rabinowitz, 1991).

TOXICOLOGY

Toxicological Effects in Animals

Acute Effects

Mean lethal dose (LD₅₀) values for lead compounds were not found in the literature, however there are lowest lethal dose (LD_{Lo}) values ranging from 20,500 mg/kg for lead sulfate in guinea pigs to 191 mg/kg for lead acetate in the dog (Sax, 1984). These are the lowest doses expected to cause death. The available data do not permit a comparison of the acute toxicity of the different lead compounds (ATSDR, 1991). Acute oral exposure of rats to lead acetate at 390 mg/kg-day caused increased fetal resorptions, retarded skeletal development and maternal toxicity (Kennedy *et al.*, 1975).

Spontaneously hypertensive Sprague-Dawley rats were given drinking water containing 100 mg/L lead as lead acetate for three weeks (Nakhoul et al., 1992). There was no change in the systolic blood pressure of the animals for the first eight days of exposure even in these genetically susceptible rats. However, by day 12 through 20, the systolic pressure of the rats was significantly higher than for the controls.

Histological changes induced by lead from lead acetate in intestines, kidney and liver were evaluated in Sprague-Dawley rats by Karmakar et al. (1986). A dose of 44 mg/kg for durations of 9, 15 or 30 days was evaluated in groups of five rats. After nine days, mild shortening of the intestinal villi was seen in two of five rats and histological changes in the liver were observed in all rats. No renal abnormalities were observed. After 15 days, intestinal and liver abnormalities had progressed and affected more animals than at nine days; three of five rats showed histological kidney abnormalities.

Chronic Effects

Numerous experiments in laboratory animals have demonstrated that lead has a wide variety of toxic effects across many different organ systems (ATSDR, 1991).

The effects of lead acetate in drinking water on the reproductive systems of male and female rats have been studied by a number of investigators. The best studies relate the oral dose to the blood lead level produced. Chowdury *et al.* (1984) observed reduced sperm counts in male rats that had blood lead levels of 72 µg/dL. No effects were observed in male rats with blood lead levels of 54 µg/dL. Both male and female rats were studied by Hildebrand *et al.* (1973). They observed irregular estrus cycles in female rats with blood lead levels of 30 µg/dL. Ovarian follicular cysts were produced in female rats with 53 µg/dL blood lead levels. They found increased prostate weight in male rats with 19 µg/dL of blood lead, and testicular damage in male rats with 30 µg/dL blood lead.

Orally administered lead acetate has been demonstrated to cause cancer in animals (Azar *et al.*, 1973). This study yielded a dose-dependent increase in the incidence of kidney tumors in rats and has been used to estimate the cancer potency of lead by the oral route (ATSDR, 1991; OEHHA, 1997).

Genetic Toxicity

Lead acetate gave conflicting results for structural chromosomal aberrations in Syrian or Chinese hamster cells (Bauchinger and Schmid, 1972; Robison *et al.*, 1984). Lead sulfide and lead nitrate were mutagenic at the hypoxanthine-guanine phosphoribosyltransferase (HPRT) locus in Chinese hamster V79 cells (Zelikoff, *et al.*, 1988). Tests of lead for mutagenicity in bacterial systems (*Salmonella typhimurium*, *Bacillus subtilis*) have yielded negative results (ATSDR, 1991).

Toxicological Effects in Humans

Acute Effects

The principal gastrointestinal effect of lead in humans is colic. This is a painful condition which involves cramps and gastrointestinal distress. Colic occurs immediately after acute exposures by

ingestion or inhalation. It corresponds to blood lead levels in the range of approximately 30 to 50 $\mu\text{g/dL}$ of blood. Colic occurs most frequently to workers who are exposed to lead in the workplace in the form of either lead-bearing dust or lead fumes from soldering or welding (Meiklejohn, 1963).

Chronic Effects

Chronic exposure to lead has been demonstrated to affect many systems of the body including the nervous system, kidneys, gastrointestinal system and the reproductive system. The effects occur at different levels of exposure, as illustrated by Figure 1 which shows the lowest level at which each of the chronic effects is observed in children.

Neurobehavioral Effects

When children or fetuses receive high doses of lead (resulting in blood lead levels near 100 $\mu\text{g/dL}$) encephalopathy may result. Lead encephalopathy is characterized by dullness, irritability, poor attention span, headache, muscular tremor, loss of memory and hallucinations. More severe cases exhibit delirium, convulsions, paralysis, coma and death (Kumar *et al.*, 1987). Children suffer encephalopathy at lower doses than adults. Encephalopathy during the 12 to 15 months after birth, during which the child's brain is developing, may lead to irreversible brain damage (Hutton, 1987; ATSDR, 1991).

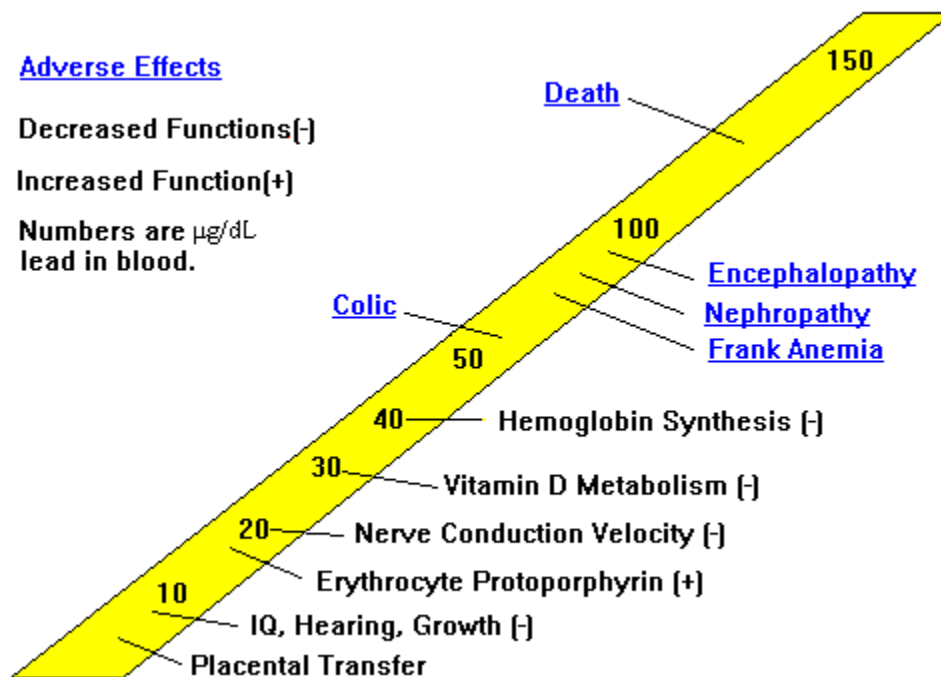


Figure 1: Lowest Demonstrated Effect Levels of Inorganic Lead in Children¹

¹ The numbers in the diagram are not necessarily the lowest levels at which lead exerts an effect. They are levels at which studies have adequately demonstrated an effect.

Neurotoxic effects at lower blood lead levels than those which cause encephalopathy (below 100 µg/dL) may in some cases result in irreversible brain damage (exhibited as a decrease in learning ability) as well as in damage to the peripheral nervous system (Hutton, 1987; ATSDR, 1991).

There have been numerous studies of the effects of low lead exposure on the intelligence of children in the U.S. and other countries. The well-known work of Needleman indicates that blood lead levels as low as 10 µg/dL may cause deficits in learning ability in very young children. Children who had umbilical cord blood lead levels at birth of 10 µg/dL or higher had poorer performance on intelligence tests and in school (Needleman, 1987). Earlier, Needleman had looked at the effect of lead on children's IQ and classroom behavior using lead levels in shed deciduous teeth as an indicator of lead exposure (Needleman, 1982). These studies showed that children with higher lead exposures had lower intelligence scores, particularly in the verbal and language-processing skills. The high-lead children also had impaired auditory processing.

A four-year follow-up of these children showed that they had poorer classroom attention than the children with less lead exposure (Needleman, 1982). Schwartz and Otto (1991) demonstrated that children with blood lead levels of 10 µg/dL had impaired hearing compared with children with blood lead levels of 6 µg/dL.

Several long-term prospective studies (for example Bellinger *et al.*, 1992; Dietrich *et al.*, 1993; and Baghurst *et al.*, 1992) have also indicated an association between post-natal blood lead and IQ in children up to 10 years of age. This suggests the persistence of the effects of lead on intelligence.

Additional studies of young children have been conducted in other countries including Australia and Scotland with similar results (Yule and Rutter, 1985; ATSDR, 1991). In Australia, a cohort in the town of Port Pirie has been studied for many years. The most recent results from this cohort indicate that there is an inverse relation between tooth lead concentration and intellectual development (McMichael *et al.*, 1994). In this case tooth lead was used as an index of cumulative exposure.

Based on these studies of IQ in children and blood lead levels from the U.S. and other countries, it appears that there is good evidence that very low blood lead levels (10 µg/dL or lower) can have a deleterious effect (a decrease of several IQ points) on the learning ability and intellectual development of young children. A decrease of only a few IQ points may be very significant on a population level in terms of increased need for remedial education (CDC, 1991).

The principal studies of the effects of lead on the learning abilities of children should be corroborated by studies in animals, but in practice this is difficult to do because animals cannot be given intelligence tests comparable to those administered to children. However, there have been studies in rodents and primates which indicate that lead exposure to neonatal or fetal animals has histological effects in the brains of these animals (Bushnell and Bowman, 1979). More recent studies in primates and rodents have demonstrated deficits in higher neurobehavioral function at blood lead levels of 15 µg/dL in primates and 20 µg/dL in rodents (Davis *et al.*, 1990).

The mechanism(s) by which lead causes neurobehavioral dysfunction in children is not known at this time. There is evidence that lead may interfere with calcium-modulated neurotransmitter pathways, disrupt the blood-brain barrier or do both (Goldstein, 1993). Lead at picomolar

concentrations has been shown to affect the function of protein kinase C isolated from rat brains (Markovac, 1988). It has been postulated that lead binds to protein kinase C at the presynaptic terminals in the brain and interferes with release of neurotransmitters (Markovac and Goldstein, 1988). Lead has been shown to enhance the neurotoxicity of certain neural proteins, which suggests the possibility that lead may harm the nervous system by causing autoimmune responses to native neural proteins (Waterman *et al.*, 1994). All of these proposed mechanisms are possible, but at this time the most significant contributors to the neurotoxicity of lead cannot be determined from the scientific information available.

Anemia and Hypertensive Effects

When lead levels are in the 50 to 100 µg/dL range, anemia may result. Anemia may be a consequence of several factors including the suppression of the heme synthesis pathway leading to shortage of hemoglobin, and the increased fragility of red blood cell membranes resulting in shorter life span of the red blood cells. The effect on the heme synthesis pathway leads to an increase of blood protoporphyrin levels. In fact these can be used as a biological monitoring method to detect lead exposure (Alessio and Foa, 1983).

Epidemiological studies indicate that increased blood lead levels result in hypertension, especially in middle aged males (Schwartz, 1991). Animal models (rats) have also shown a relationship between blood lead and hypertension. Hypertension may also be caused in females or other age groups, but it has been most extensively studied in middle aged males. The blood lead level at which this effect appears to begin is approximately 10 µg/dL (Schwartz, 1991). Therefore, the lowest-observed-adverse-effect-level (LOAEL) for this effect is approximately the same as that for decreased IQ in children. It therefore seems prudent to regard 10 µg/dL as a minimum toxic level for both children and adults.

Hypertension or “high blood pressure” increases the risk of cardiovascular diseases leading to heart attacks or strokes (Schwartz, 1991; Pirkle *et al.*, 1985). Therefore, an agent which increases the mean blood pressure would have an effect on mortality in a population, increasing mortality and decreasing life expectancy. The number of years of life lost can be calculated, since a relationship can be established between blood lead levels and hypertension (Schwartz, 1991; Pirkle *et al.*, 1985; Harlan *et al.*, 1985).

Kidney Effects

Lead exposure, at doses intermediate between those that cause intelligence deficits and those that lead to encephalopathy, may result in nephropathy, manifested as impaired kidney function and the presence of glucose, amino acids and other biochemicals in the urine (Fowler and DuVal, 1991). This effect has been demonstrated in humans and animals. The mechanism involves structural changes in the kidney tissue which lead to blockage of the kidney tubules (Fowler and DuVal, 1991).

Reproductive Effects

The effects of lead acetate in drinking water on the reproductive systems of male and female rats have been studied by a number of investigators. The best studies relate the oral dose to the blood lead levels produced. Chowdhury *et al.* (1984) observed reduced sperm counts in male rats that had blood lead levels of 72 µg/dL. No effects were observed in male rats with blood lead levels of

54 µg/dL. Both male and female rats were studied by Hildebrand *et al.* (1973). They observed irregular estrus cycles in female rats with blood lead levels of 30 µg/dL. Ovarian follicular cysts were produced in female rats with 53 µg/dL blood lead levels. They found increased prostate weight in male rats with 19 µg/dL of blood lead, and testicular damage in male rats with 30 µg/dL.

Lead has long been used as an abortifacient in humans (Meiklejohn, 1963). Women who worked in the ceramics industry in Britain around the end of the 19th century appeared to have an increased incidence of stillbirths due to accidental or deliberate ingestion of the lead glaze material (Meiklejohn, 1963).

Cancer

Orally administered lead acetate has been demonstrated to cause cancer in animals (i.e., it increased the incidence of kidney tumors in rats) (Azar *et al.*, 1973). This study has been used as the basis for estimating the cancer potency of lead (ATSDR, 1991; OEHHA, 1997). Lead is regarded by the International Agency for Research on Cancer (IARC) and the U.S. Environmental Protection Agency (U.S. EPA) as an animal carcinogen and probable human carcinogen (IARC, 1987; U.S. EPA, 1989). Given that lead acetate is carcinogenic in rats (Azar *et al.*, 1973), other ionic salts would probably be carcinogenic as well.

Summary of Chronic Health Effects in Humans

The most significant health effects from the public health and regulatory point of view are the ones which occur at the lowest blood lead levels, because these affect the greatest part of the population. For children these are the effects on intelligence and behavior. For adults the critical health effect is the increase in blood pressure. Both of these health effects are of concern in the vicinity of approximately 10 µg/dL blood lead. As both of the critical health effects for lead may occur near 10 µg/dL, this level may be considered a level of concern for both children and adults. Other health effects such as kidney and gastrointestinal effects occur at higher blood lead levels. See Figure 1 for a summary of these effects and the blood lead levels at which they occur.

DOSE-RESPONSE ASSESSMENT

Noncarcinogenic Effects

The critical health end points for lead are intelligence deficits in children, and hypertension in adults. The PHG is developed based on intelligence deficits in children, as this is the best documented health end point that occurs at very low levels of exposure, and because it results in a lower PHG than the hypertension end point.

Based on studies correlating blood lead levels with decreased IQ in children, the Centers for Disease Control (CDC) has identified 10 µg/dL (blood) as the lowest blood lead level of concern (CDC, 1991). Using an IEUBK model (Version 0.99d, 1994), OEHHA determined that for children between 12 and 24 months a blood lead level increase of 0.35 µg/dL results from each increment in drinking water intake of 1.0 µg/day (OEHHA, 1997). This was based on a calculation using the default values for exposure from dust, air, paint and other sources.

Therefore, the lead intake level that would correspond to the level of concern for children can be calculated as follows:

$$\begin{aligned}\text{Lead intake} &= \frac{10 \mu\text{g/dL (blood)}}{0.35 \mu\text{g/dL per } \mu\text{g/day}} \\ &= 28.6 \mu\text{g/day}\end{aligned}$$

This is the daily lead intake that corresponds to a blood lead level of 10 $\mu\text{g/dL}$. In other words, 28.6 $\mu\text{g/day}$ can be used as a benchmark for daily oral intake from water that corresponds to a level of concern for neurological effects in children.

Carcinogenic Effects

The best study for assessment of the carcinogenic effects of lead by the oral route is the study by Azar *et al.* (1973). In this experiment rats were administered lead acetate in their diet. Kidney tumors were produced in a dose-related manner. Table 1 shows the data from this experiment.

Table 1. Kidney Tumor Incidence in Rats Administered Lead Acetate in the Diet (Azar *et al.* 1973).

Dose (mg/kg-day)	Number of Rats in Dose Group	Number of Rats with Tumors
0.23	20	0
0.39	100	0
1.40	50	0
4.78	50	0
10.9	50	0
42.3*	20	5
79.7*	20	10
167*	20	16

*The groups with only 20 rats per dose group were also studied for two years but were begun several months after the other dose groups.

From these data, a q_1^* (animal) was calculated using Global 86. The p value for the Chi Square test was 0.82, indicating a good fit of the data to the linearized multistage model. The q_1^* (animal) is $1.53 \times 10^{-3} (\text{mg/kg-day})^{-1}$. An equivalent human q_1^* was calculated using the formula

$$\begin{aligned}q_1^* (\text{human}) &= q_1^* (\text{animal}) \times (\text{human body weight/rat body weight})^{0.25} \\ &= 1.53 \times 10^{-3} (\text{mg/kg-day})^{-1} \times (70 \text{ kg}/0.3 \text{ kg})^{0.25} \\ &= 5.98 \times 10^{-3} (\text{mg/kg-d})^{-1}\end{aligned}$$

An oral cancer slope factor (CSF) was calculated using the formula:

$$\text{CSF} = 0.1 \div \text{LED}_{10}$$

The LED₁₀ is the 95% lower-bound on dose resulting in a 10% tumor incidence as calculated by, for example, Global 86. For the rat kidney tumor data, the LED₁₀ is 68.8 mg/kg-day, and the CSF (rat) is $0.1 \div 68.8 \text{ mg/kg-day} = 1.45 \times 10^{-3} (\text{mg/kg-day})^{-1}$.

The CSF for the rat data was converted to a CSF for humans using the same body weight scaling as described for the q₁*. This calculation yielded a CSF (human) of $5.68 \times 10^{-3} (\text{mg/kg-day})^{-1}$. Therefore, the CSF (human) is approximately the same as the q₁* (human). This CSF (human) was used to calculate a PHG based on carcinogenicity, as described below.

CALCULATION OF PHG

Carcinogenic Endpoint

A public health-protective concentration (C) for lead (in mg/L) in drinking water can be calculated using the general equation for carcinogenic endpoints:

$$C = \frac{R \times BW}{CSF \times L/\text{day}} = \text{mg/L}$$

where,

R	=	<i>De minimis</i> theoretical excess individual lifetime cancer risk value of 1×10^{-6}
BW	=	Adult male body weight default value (70 kg)
CSF	=	Cancer slope factor calculated previously [$5.68 \times 10^{-3} (\text{mg/kg-day})^{-1}$]
L/day	=	Volume of daily water consumption for an adult (2 L/day).

Therefore,

$$\begin{aligned} C &= \frac{1 \times 10^{-6} \times 70 \text{ kg}}{5.68 \times 10^{-3} (\text{mg/kg-day})^{-1} \times 2 \text{ L/day}} \\ &= 6.16 \times 10^{-3} \text{ mg/L} = 0.006 \text{ mg/L (rounded)} = 6 \text{ ppb.} \end{aligned}$$

Noncarcinogenic Endpoints

A public health-protective concentration (C) for lead (in mg/L) in drinking water can also be calculated using the general equation for noncarcinogenic endpoints:

$$C = \frac{NOAEL \times RSC}{UF \times L/\text{day}} = \text{mg/L}$$

where,

NOAEL	=	No-observed-adverse-effect-level or for lead is substituted with the “level of concern” for children (28.6 µg/day)
RSC	=	Relative source contribution of 20% (0.2)
UF	=	Uncertainty factor of 3-fold
L/day	=	Daily volume of consumption of drinking water for a child (1 L/day).

The NOAEL is substituted in this equation by a level of concern of 28.6 µg/day derived previously for children. CDC's level of concern for lead in blood is somewhat arbitrary, but has been consistently lowered over the last two decades, and may be lowered again in the future. There is some uncertainty as to whether this level is protective for all children. There are children in the population whose blood lead levels are already above the concern level. For these individuals any increase in blood lead level would simply add to an already adverse blood lead level. In calculating the PHG for noncarcinogenic effects, an uncertainty factor of three was applied to account for the uncertainty with regard to the degree of protection offered by the estimated level of concern, because no threshold has been observed for the noncarcinogenic effects of lead and there is a wide distribution of blood lead levels in the population of children (African-American children have higher levels than the general population).

For purposes of the PHG calculation, children are assumed to consume 1 L of water/day. The drinking water contribution to children's lead exposure is estimated to range from 5% to over 50% (U.S. EPA, 1991) depending on the immediate environment in which the child lives. For children exposed to lead in paint, or lead in air and soil (e.g., living near roadways where lead deposits from engine exhaust still persist), U.S. EPA determined that drinking water exposure to lead would be on the lower end of this range. Therefore, in calculating a public health-protective concentration, we assume that drinking water exposures would contribute 20% of the total exposure to lead to account for exposures in children living in areas where high environmental concentrations of lead still persist.

Therefore,

$$\begin{aligned} C &= \frac{28.6 \mu\text{g/day} \times 0.2}{3 \times 1 \text{ L/day}} \\ &= 0.0019 \text{ mg/L} = 0.002 \text{ mg/L (rounded)} = 2 \text{ ppb.} \end{aligned}$$

The public health-protective concentration for lead based on the carcinogenic endpoint is 6 ppb. This is higher than the public health-protective concentration of 2 ppb calculated for noncarcinogenic effects. Therefore, the Public Health Goal (PHG) developed for lead in drinking water is 0.002 mg/L (2 ppb) based on noncarcinogenic effects.

RISK CHARACTERIZATION

The health risks of exposure to lead are well established by a large body of research. For the noncarcinogenic effects upon which this PHG is based (i.e., neurobehavioral effects to children and hypertensive effects to adults), the research has been conducted on human populations. Therefore, there is no uncertainty based on extrapolation from animals to humans for these effects. The carcinogenic effect data is based on animal experimentation. In assessing the risk from carcinogenicity, there is uncertainty in extrapolating from animals to humans. The Azar *et al.* (1973) study is the best available data for calculating a CSF.

Humans, especially children, may vary in their sensitivity to lead in drinking water because of differences in nutrition, exposure to lead from other sources and metabolic and genetic differences. Adults may vary in their sensitivity to the hypertensive effects of lead.

The calculated PHG utilizes an RSC of 20% (0.2). This value is justified for certain subpopulations of children living in areas where lead in the environment still persists in moderate to high levels. Higher RSCs (up to 50%) might be justified for the general population because of the recent declines in relative contribution from air, water and food. The use of a higher RSC would increase the calculated PHG for noncarcinogenic endpoints for lead in drinking water.

OTHER STANDARDS AND REGULATORY LEVELS

U.S. EPA has adopted a Maximum Contaminant Level Goal (MCLG) of zero for lead in drinking water, based on “occurrence of low level effects” and because U.S. EPA classifies lead as a Class B₂ carcinogen (U.S. EPA, 1997; Fed. Reg. 56: 32112, July 15, 1991). U.S. EPA has not adopted a Maximum Contaminant Level (MCL) for lead in drinking water because they regard the development of such a level as “not feasible” (U.S. EPA, 1997; Fed. Reg. 56: 32112, July 15, 1991). U.S. EPA relies on the “treatment approach” described in the final rule (Fed. Reg. 56: 32112, July 15, 1991) to achieve the objective of reducing exposures to lead. U.S. EPA has also set an “action level” for lead in drinking water of 15 ppb (40 CFR 141, 142; Fed. Reg. 56: 26461-26564). This is a level the U.S. EPA believes is feasible for public water systems to attain by such measures as adjusting the physical characteristics of the water (pH, hardness) which affect the corrosivity of the water. U.S. EPA has set a National Ambient Air Quality Standard of 1.5 µg/m³ (Fed.Reg. 43: 41258, October 5, 1978).

The lead and copper rule is a Federal and State drinking water standard (Title 22 CCR, section 64672.3) that specifies requirements for lead in drinking water systems (measured at the customers’ taps). The action level (15 ppb) is used to determine the treatment requirements that a water system must complete. The action level for lead is exceeded if the concentration of lead in more than 10 percent of the tap water samples collected during any monitoring period (conducted in accordance with 22 CCR sections 64682 to 64685) is greater than 15 ppb. Failure to comply with the applicable requirements for lead and copper is a violation of primary drinking water standards for these substances (22 CCR Chapter 17.5). Therefore, for all practical purposes the standard described in the lead and copper rule is an MCL.

U.S. EPA has set a National Ambient Air Quality Standard of 1.5 µg/m³ (Fed. Reg. 43: 41258, October 5, 1978).

Lead is listed as a carcinogen and as a reproductive and developmental toxic chemical under the Safe Drinking Water and Toxic Enforcement Act of 1986, “Proposition 65” (California Health and Safety Code, Chapter 6.6, section 25249.5 *et seq.*). Lead is listed as a reproductive and developmental toxic chemical because of its effects on IQ during development. Under this program the level set for warning against possible reproductive and developmental effects is 0.5 µg/day for any one source of exposure.

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